ABDOMINAL VISCERAL SENSATION IN MAN*

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THE RECOGNITION of the existence of visceral pain has been comparatively recent. Before and for some years after Langley's[1] (1903) review of the functions of the autonomic nervous system it was believed that such afferent fibers as were known to accompany autonomic nerves served only in reflex mechanisms to autonomic tissues and were "incapable of directly giving rise to sensation." Ross[2] in 1887 had shown that tension in a hollow viscus produced pain that was perceived in a corresponding cutaneous dermatome on the abdominal wall. But Lennander[3] (1902) as a result of observing the lack of pain when the bowel was cut or otherwise traumatized concluded that the bowel was insensitive to painful stimuli and that any pain accompanying visceral stimulation resulted from irritation of the pain sensitive parietal peritoneum. It was not until 1911 that Hurst,[4] employing balloon distention of the bowel, demonstrated conclusively in man that sensation from the bowel could be elicited and emphasized the need for employing an adequate stimulus in studying visceral sensation.

Stimulation of the parietal pleura or peritoneum by friction or faradism results in pain, the impulses travelling via somatic afferent fibers in spinal nerves. But visceral pleura and peritoneum have been found insensitive to similar stimuli, and most observers have likewise found the mucosa of the stomach and bowel to lack pain sensitivity.[5, 6, 7] The stimulus adequate for the production of visceral pain is believed by many to be traction on the mesenteries, and Alvarez[6] states that neither distention nor contraction of a hollow viscus causes pain unless the adjacent mesentery is stretched. On the contrary, others[8, 9] believe that pain which results from distention of a viscus is due to stretching of its sensory nerves, while Hurst[4] and Ryle[10] contend that tension of the muscular coat or a distended or contracted viscus provides the stimulus for pain.

Many analogies can be drawn between somatic and visceral pain but certain striking differences are important to recognize for purposes of clinical investigation. Somatic pain which is conducted by spinal nerves has the property of sharpness and accurate surface localization which is largely acquired after birth, while visceral pain on the contrary is deep, dull and less well localized. But sometimes there may be a supplementary mechanism by which visceral pain is referred to a cutaneous region which has a corresponding segmental innervation. Sometimes this referred pain has been looked upon as the sole manifestation of visceral pain but blocking of the painful area locally

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or interruption of the spinal nerves supplying the area by procainization cannot be counted on to abolish visceral pain.[11]

The matter of the types of end organ for visceral pain reception and their exact location is still unsettled. Tyrrell-Gray[12] (1922) advanced the view that the afferent end organs were the Pacinian corpuscles occupying the mesentery and that they responded to mechanical, thermal, chemical and electrical stimuli. Sheehan[13] (1933) summarized his studies of degeneration following section of the vagi and sympathetics by concluding that visceral pain in the abdomen arises from mesenteric nerve endings of 3 types: (1) Pacinian Bodies, (2) free endings of myelinated nerves and, (3) free endings of a fine plexus of non-myelinated fibers. The Pacinian corpuscles were found to be distributed to the mesentery of the bowel chiefly around large vessels but not in the bowel wall, and in the pancreas and about the biliary tree but not in the omentum. Splanchnicectomy resulted in degeneration of the Pacinian corpuscles.

The vagus nerves possess afferent fibers which are believed to conduct certain reflex stimuli from the abdominal viscera but not pain sensation (Cannon[14]). While some still doubt that the vagus nerves do not transmit pain impulses from abdominal viscera almost all investigators agree that the splanchnics and other sympathetic nerves do possess visceral pain fibers. The axones of the visceral afferent nerves accompanying the sympathetic nerves are poorly myelinated and their rate of conduction is slow. They run all the way to the cord without synapse and enter via the posterior spinal roots. Earlier contention that visceral afferent impulses traversed anterior spinal roots[15] has been largely discredited by later work such as that of Davis, Pollock and Stone[11] which concluded that visceral afferents enter the cord only by the posterior roots.

A table of the segmental sensory innervation of the viscera was compiled by White and Smithwick[16] (1941) from available experimental and clinical data, but much of the evidence is of necessity inferential. On the whole, comparatively few observations have been made in man on the pathways for the conduction of visceral pain. Efforts to map the spinal segments which transmit painful sensations from various levels in the gastro-intestinal tract by paravertebral injection of anesthetic agents have been described by Läwen[17] and by Kappis and Gerlach.[18] Adson[19] (1935) and Leriche[20] (1937) stimulated the splanchnic nerves in the course of operations performed during spinal anesthesia and produced pain in the upper scapular region on stimulation of the greater splanchnic, and pain lower down on stimulation of the lesser nerve. White[21] (1943) reviewed the sensory innervation of the viscera in man based on neurosurgical procedures performed for the relief of intractable pain and added an account of his own experiences.

Conclusions drawn from the results of somewhat blind injections of anesthetic agents about unseen nerves usually leave some doubt and the aggregate of experiences with alteration in visceral sensation after surgical interruption of visceral afferent nerves is relatively meager and inconclusive. For example, pain sensation originating in the heart and in the urinary bladder has received

greater attention than almost any other form of visceral sensation, yet the information is incomplete. Sympathectomy[16, 22] and posterior rhizotomy[23, 24] for relief of cardiac pain have effectively demonstrated afferent pathways through the upper 5 thoracic sympathetic ganglia and posterior roots but leave unexplained the residual pain in the neck which occurs in some patients. Learmonth's[25] investigations of bladder sensation indicate the role of visceral afferent fibers in the triple innervation of the bladder but leave unanswered the detailed pathway of the sensory fibers accompanying the sympathetic nerves.

The present study was carried out on a series of patients most of whom had hypertensive cardiovascular disease and all of whom were subjected to thoracolumbar sympathectomy in which the paravertebral ganglionated chain was resected from the seventh thoracic through the third lumbar ganglia, and the splanchnics were resected from the celiac ganglia to a point above the contribution of the seventh thoracic rami. In certain studies additional interruption of nerve pathways supplemented the standard sympathectomy. Observations were made on the pain sensitivity of the stomach, large and small intestine, biliary tree, pancreas and urinary tract prior to denervation, and at various times up to one year after right or left sympathectomy and after bilateral sympathectomy. Such tests are tedious and try the patience of both the subject and the examiner. The use of the tests and interpretation of the results require circumspection. All of the results to be reported have been observed more than once and in some cases many times, and while it is realized that additional observations are desirable the findings seem adequate at least for a preliminary report.

BALLOON DISTENTION OF THE INTESTINAL TRACT

Fifteen patients were selected for this study. A rubber bag attached to the end of a Miller-Abbott tube was passed through the mouth to various levels in the intestinal tract and the position of the bag identified by fluoroscopy.

The main cavity of the stomach because of its size and the distensibility of its wall does not lend itself readily to studies with the balloon and therefore no observations of pain by distention were made. However, the effects of temperature were studied (see section on THERMAL STIMULATION).

The *small bowel* was distended at numerous points from the duodenal cap to the cecum (Fig. 1). As a rule the pain thus induced was normally felt in a relatively discrete area somewhere in the midline between the xiphoid and the umbilicus; that from the duodenum was felt in the upper epigastrium, and that from the jejunum and ileum nearer the umbilicus. A volume of 50 to 75 cc. of air in the balloon producing a pressure of 1 to 1.5 centimeters of mercury was sufficient to cause a distinct but bearable deep aching pain. Distention to 90 to 120 cc. of air at a pressure of 1.5 to 2 cm. of mercury was perceived as a moderate to severe pain, while more than 120 cc. of air at 3 cm. mercury pressure caused severe and more widespread pain in the midline of the abdomen. Patients subjected to the latter pressure often became pallid,

developed a cold, clammy feeling and a desire to eructate. Pain did not occur in the back. While distention of the jejunum and upper ileum was always accompanied by pain at or above the umbilicus distention of the terminal ileum sometimes also caused pain below the umbilicus and occasionally about McBurney's point. This exceptional pain below and to the right of the umbilicus

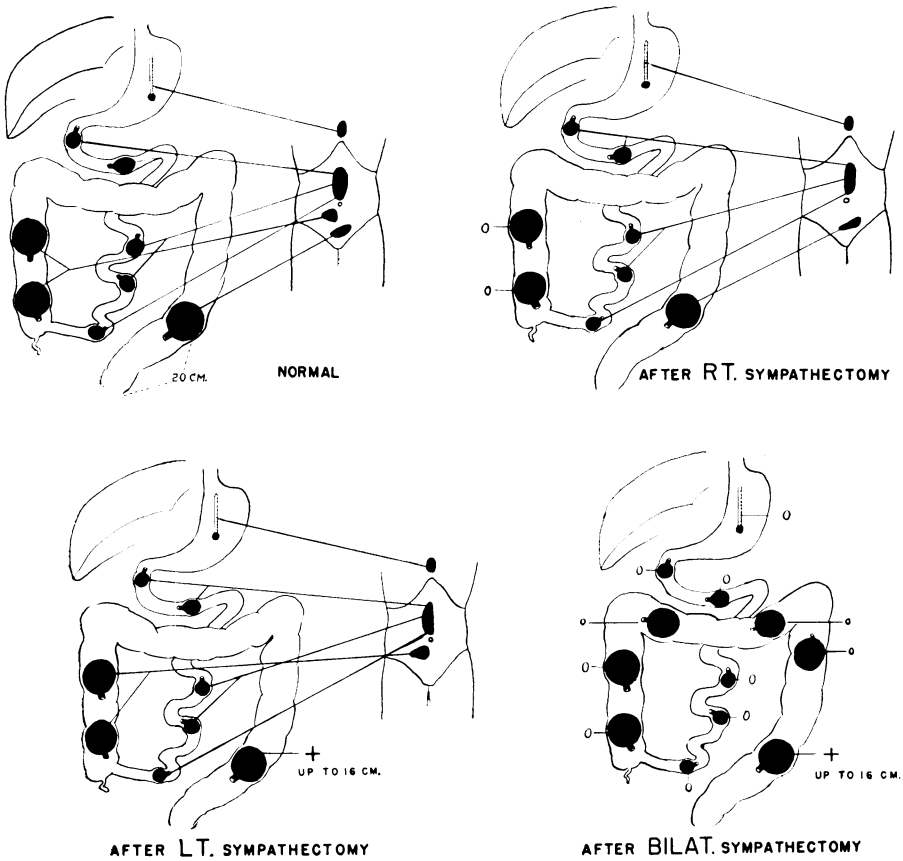

FIG. 1.—Balloon distention of gastro-intestinal tract. Eight areas of sensory reference.

did not have the deep aching character of the usual para-umbilical pain and could have occurred as a result of local parietal peritoneal stimulation or as a referred pain.

On repetition of the tests after either left or right sympathectomy, there was no alteration in the region at which pain was felt or in the threshold of

pain appreciation. There was no tendency for lateralization of the pain to the undenervated side. After bilateral sympathectomy pain could not be elicited with distention even after 300 cc. of air at 4 to 6 cm. of mercury pressure.

The *large bowel* is less satisfactory to investigate because of the difficult and unpleasant task of passing the tube and balloon the longer distance and maintaining it for the period of time necessary for the tests. However, enough observations were made to afford general conclusions. About the same pressure within the balloon sufficient to cause pain in the small bowel was adequate stimulus for pain in the large bowel with the possible exception that relatively higher pressure was sometimes needed in the cecum to cause pain of comparable degree to that from 1 to 2 cm. mercury pressure in the rest of the large bowel. The sites of pain were relatively discrete and became more diffuse only with increased pressure within the balloon. The patterns of reference are indicated in Figure 1. Normally, distention of the cecum was perceived as pain in an area just below and to the right of the umbilicus and pain arising from the hepatic flexure and proximal portion of the transverse colon occurred at points within a larger area extending from the umbilicus downward and to the right. From distention of the splenic flexure and descending colon pain was felt in the lower abdomen to the left of the umbilicus, while pain from the rectosigmoid was distributed across the suprapubic region extending perhaps more to the left side. In addition to tests with the balloon passed orally, several satisfactory observations on the lower bowel in its last 50 cm. were made using a tube with a coil spring incorporated in a #20 French catheter passed through a proctoscope. (This tube was devised by Thomas P. Almy and Fred Kern of the New York Hospital.)

After right sympathectomy the only alteration in large bowel sensation observed was absence of pain on distention of the cecum. After left sympathectomy no alteration in large bowel sensation resulted from the studies employed although in the patients examined no satisfactory observations were made of distention of the splenic flexure and descending colon. After bilateral sympathectomy the appreciation of painful distention of the large bowel was abolished above the level of 16 cm. from the anus. Below this level, distention of the bowel with the minimal adequate pressure still elicited pain in the suprapubic region. In addition, distention of this lowest segment produced a desire to defecate.

THERMAL STIMULATION

In normal subjects distention of a balloon in the stomach with water at temperatures of 110° to 120° F. caused a feeling of heat in the subxiphoid region, while water at 32° F. caused a feeling of cold in the same region. Water temperatures between these extremes were not definitely perceived and one subject did not appreciate either hot or cold. In 8 of 9 subjects examined after bilateral sympathectomy the ability to perceive temperature in the

stomach was abolished. In the ninth it persisted, but the area of reference before and after sympathectomy was at the sternal notch at the base of the neck. A few additional observations on thermal stimulation of the small bowel suggested that extremes of temperature are not perceived there but the evidence was incomplete.

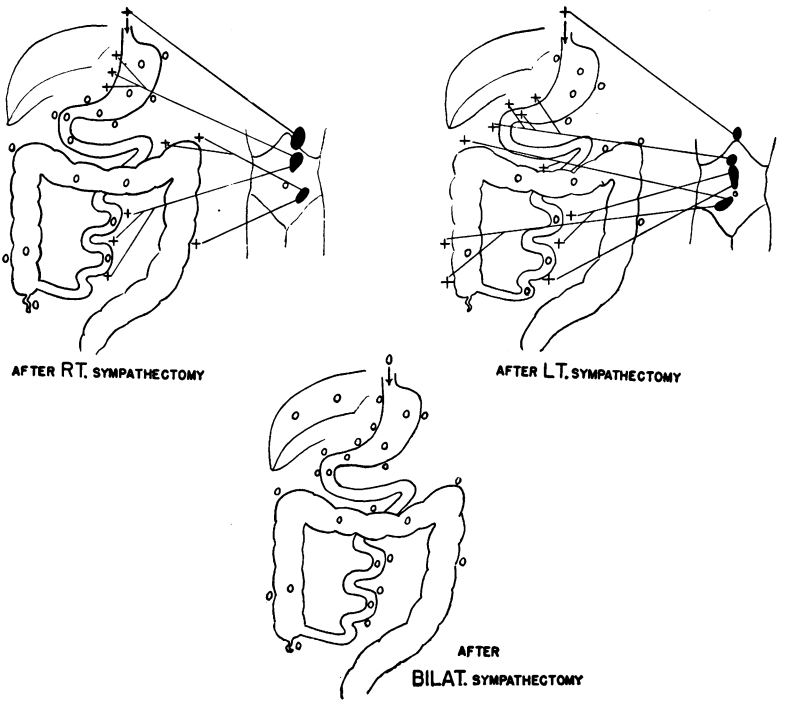

FIG. 2

FIG. 2.—Stimulation of gastro-intestinal tract (faradic, traction stretch) through celiotomy.

DIRECT STIMULATION OF THE STOMACH AND INTESTINAL TRACT THROUGH CELIOTOMY

The combined observations were made on 6 patients having unilateral or bilateral sympathectomy in whom some intra-abdominal operation was necessary. Regional procain anesthesia was employed only to open the anterior abdominal wall and peritoneum. The methods of stimulation employed included pinching and local stretching of the gastric and intestinal walls, traction on the mesenteries and omentum, and faradic stimulation of various structures. The electrical stimulation was performed with the Hinsey-Geohegan stimulator using a bipolar electrode and a current of between 3 to 5 volts. The results are diagrammed in Figure 2.

Even though tests were not made on unsympathectomized patients some

part of the gastro-intestinal tract was pain sensitive in the combined group of patients having unilateral (either right or left) sympathectomy. Pinching, local stretching, and faradic stimulation of the walls of the stomach and intestine failed to cause pain except at points close to the proximal mesenteric attachments. The greater curvature of the stomach, the gastrocolic omentum, and the greater omentum as well as the larger vessels they contain were not found to be pain sensitive. When any pain sensitivity was elicited along the gastro-intestinal tract it resulted from some form of stimulation of the mesentery or at the mesenteric attachment to the viscus.

The stomach. After right sympathectomy, traction on the cardiac end of the stomach produced pain first in the left upper abdominal quadrant. As the traction was increased precordial pain, a "tugging sensation in the gullet" and nausea were added to the abdominal pain. Traction and electrical stimulation applied to the mesentery and border of the lesser curvature of the stomach along the proximal half of its extent also resulted in pain in the left upper abdomen. Similar stimulation of the pylorus and first portion of the duodenum did not elicit pain. The reverse was found in patients after left sympathectomy, *i.e.,* the proximal half of the lesser curvature and its mesentery were insensitive while the pylorus and first portion of the duodenum were sensitive, the site of reference of the pain being in a fairly discrete area in the midline and possibly slightly to the right in the epigastrium. After bilateral sympathectomy all these positive responses of pain were abolished.

The jejunum and ileum. After unilateral sympathectomy, traction and, less constantly, electrical stimulation of the mesentery of the small bowel below the ligament of Treitz always resulted in pain in a relatively localized area above the umbilicus. After right sympathectomy the pain elicited was to the left of the midline, while after left sympathectomy the pain was in the midline or possibly slightly to the right of it. After bilateral sympathectomy no pain could be elicited from any form of stimulation of the small bowel or its mesentery.

The large bowel. After right sympathectomy traction and, less constantly, electrical stimulation of the mesentery or adjacent portion of the peritoneum (as in the case of a retroperitoneal cecum) of the colon as far as the middle of the transverse colon failed to elicit pain. The same was true of the appendix and its mesentery. But traction of the mesentery at the splenic flexure and of the descending colon (only the upper portion was examined) resulted in pain in the right lower quadrant of the abdomen. The reverse was true after left sympathectomy in that no pain followed traction on the mesentery of the left half of the colon above the pelvic brim while traction and, sometimes electrical stimulation of the mesentery of the appendix, cecum, hepatic flexure, and right half of the transverse colon elicited pain in the right lower quadrant. After bilateral sympathectomy repetition of the tests failed to produce pain when applied to any part of the colon or its mesentery above the sigmoid.

TESTS OF PAIN SENSITIVITY OF THE BILIARY TRACT AND PANCREAS (FIGURE 3)

The tests were made through the open abdomen on 6 patients who had had unilateral or bilateral sympathectomy and in whom celiotomy was necessitated because of chronic disease of the gallbladder, stomach or pancreas. In addition, observations on several unsympathectomized patients were made, sufficient to establish the adequacy of the tests and to confirm similar studies made by others on normal sensation of the biliary tract. Regional procain

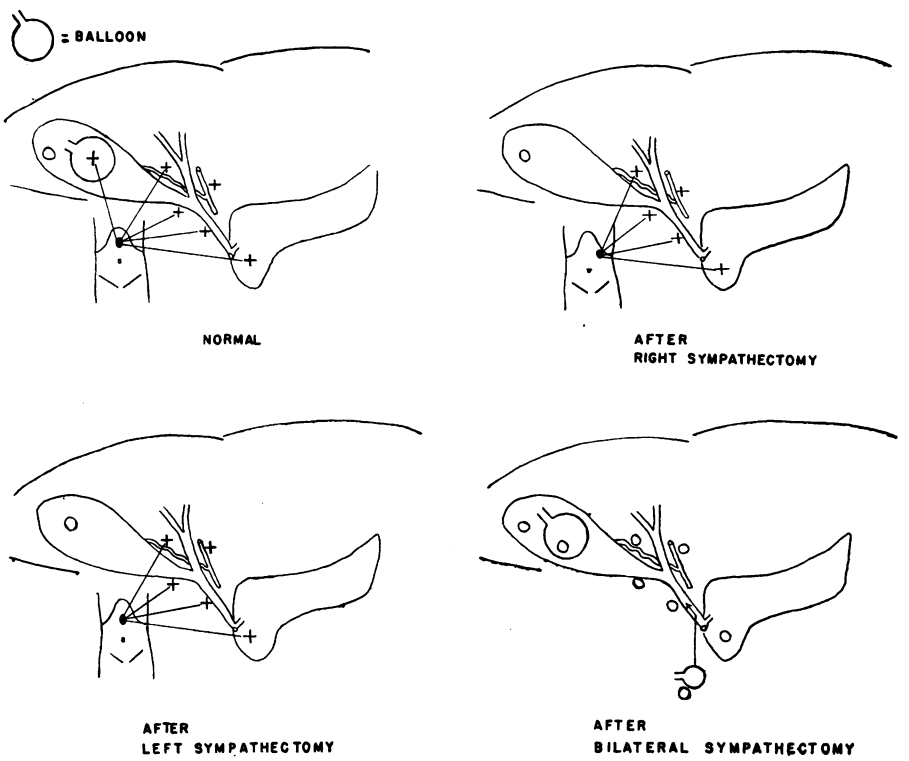

FIG. 3.—Stimulation of biliary system and pancreas stretch, faradic and distention.

anesthesia was employed to open the anterior abdominal wall and peritoneum. The methods of stimulation employed included balloon distention of the gallbladder, distention of the common duct by saline injection, and traction and faradic stimulation (3–5 volts) of all of the extra-hepatic biliary tree, the cystic vessels and the pancreas.

In the unsympathectomized patients distention of the gallbladder by inflation of the balloon with air at 4 cm. mercury pressure caused pain in a relatively small area in the midline of the epigastrium. The fundus of the

gallbladder and the capsule of the exposed liver were not pain sensitive to manipulation, local traction, or electrical stimulation. But these stimuli applied to the cystic duct, common duct, cystic artery, or head of the pancreas, each produced pain in the same spot in the mid-epigastrium. Pain did not occur in the back or in the hypochondrium.

After right sympathectomy there was no opportunity to study the effects of balloon distention of the gallbladder, but repetition of all the other tests on the biliary tract, the vessels, and head of the pancreas caused pain in the left hypochondrium. After left sympathectomy the same structures were found to be pain sensitive, but the pain was experienced in the mid-epigastrium; in addition, the pain from pressure on the head of the pancreas extended to the right hypochondrium and that from stimulation of the cystic and common ducts in one patient was experienced not only in the epigastrium but in the right infrascapular region. After bilateral sympathectomy no pain could be elicited on repetition of the tests even though the intensity of the stimuli was increased.

TESTS OF PAIN SENSITIVITY OF THE UPPER URINARY TRACT (FIGURE 4)

In 15 patients cystoscopic examinations before and after unilateral or bilateral sympathectomy permitted observations on pain sensitivity of the ureter. In the unsympathectomized subject mechanical irritation and faradic stimulation (3 volts) at the ureteral orifice and in the distal 6 cm. of the ureter caused pain over and slightly above the symphysis pubis. Rapid injection of 10 to 20 cc. of saline into the ureter (and kidney pelvis) through a #8 French ureteral catheter was very painful, the pain occurring in a broad area in the lower half of the abdomen and flank on the side stimulated. After unilateral sympathectomy the normal responses were abolished on the denervated side; there were no contralateral changes.

Two hypertensive patients undergoing sympathectomy without general anesthesia were suitably intelligent and cooperative to permit tests on the exposed kidney and upper half of the ureter. Regional procain block was used in the operative exposure of these structures and care was taken not to procainize any of the intercostal or sympathetic nerves. Faradic stimulation (up to 5 volts) of the renal capsule and pressure on the kidney at points not bordering on the renal pelvis were not painful. However pain occurred in a localized area at about the outer tip of the 12th rib on the same side when the renal pelvis, the pedicle, or the ureter near the uretero-pelvic juncture were stretched or compressed. The same type and location of pain followed faradic stimulation of these structures, including the isolated renal artery and vein. Traction and faradic stimulation of the ureter at points distal to the first several centimeters caused pain in the inguinal region on the same side. Division of the greater and lesser splanchnic nerves at the celiac ganglion and resection of the thoracic ganglionated chain from Th7 to 11, inclusive resulted in elimination of pain responses when the tests on the renal pelvis and pedicle were repeated. The pain caused

by stimulation of the ureter was not eliminated until Th12 and L1 ganglia were excised, thus extending the resection of the sympathetic chain to a point below the first lumbar ganglion.

TESTS OF PAIN AND THERMAL SENSITIVITY OF THE URINARY BLADDER

Observations on bladder sensation in several unsympathectomized patients were made in order to establish the adequacy of the tests and confirm

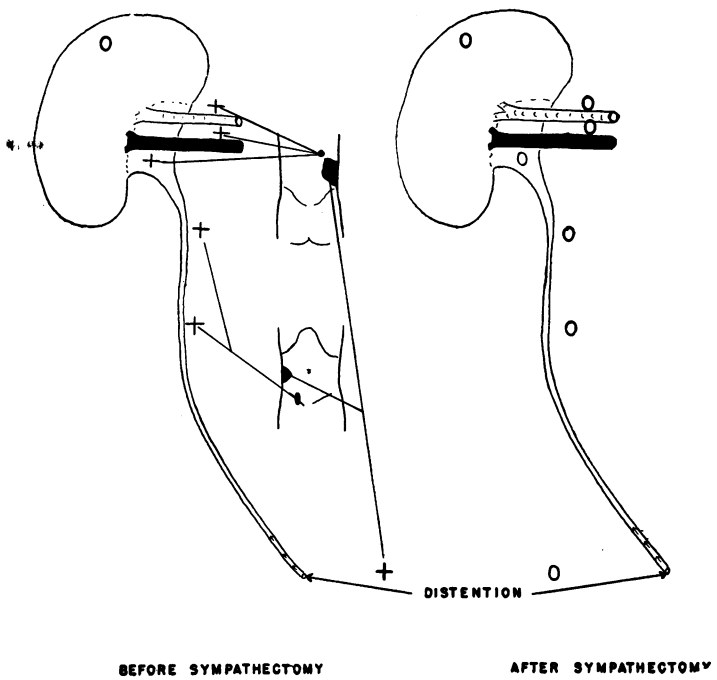

FIG. 4

FIG. 4.—Stimulation kidney, ureter and renal vessels and areas where pain is felt.

previous studies on normal bladder sensation. Faradic stimulation to the trigone (1 volt) and to the dome (2–3 volts) caused pain in the suprapubic region and over distention of the entire bladder caused similar pain. Installation in the bladder of water at 110° F. was detected as warmth in the region of the pubis while water at 65° F. or lower was perceived as cold in the same region. After right or left unilateral and bilateral sympathectomy, there was no appreciable change in these observations. The cystometric curve and bladder capacity were not altered from the normal after bilateral sympathectomy.

Additional light was thrown on the source of sensory supply to the bladder by studies on 2 patients who had had all sacral nerve roots (motor

and sensory) cut for relief of intractable pain due to malignant chordoma invading the sacrum. The bladder sensations resulting from faradic and thermal stimuli appeared normal both before and after the rhizotomies. There was urinary incontinence preoperatively and in both patients before and after rhizotomy there was a sense of suprapubic fullness after filling the bladder with 200 cc. of water and of pain after 300 cc. or more. In these patients bilateral lumbar sympathetic blocks were performed by injecting procain paravertebrally and a repetition of the tests showed that all sensation in the bladder was eliminated except a vague feeling of distress when the bladder was distended by 500 cc. or more of fluid.

Since, in the two patients subjected to complete sacral rhizotomy, the visceral afferents of the bladder accompanying the parasympathetic nerves (S2, 3, 4) and the somatic sensory fibers to the pudendal nerves (S3 and 4) were all divided, no distinction between the role of these two groups of nerves was made. However, when the pudendal nerves were blocked in the perineum by procainization in patients previously subjected to bilateral sympathectomy normal bladder responses to the standard stimuli still existed. In these patients it was assumed that the only fibers remaining to transmit sensory impulses were the visceral afferents (S2, 3, 4) accompanying the parasympathetics.

DISCUSSION

In determining any aspect of human visceral pain man has distinct advantages as an experimental animal. The opportunity for the present study was ideally provided by a selected group from several hundred hypertensive patients subjected to sympathectomy for their disease. Since the extent of the operation was believed to interrupt all or the majority of sensory nerves thought to contribute to abdominal visceral sensation, it was not unexpected to find that bilateral sympathectomy from Th7 to L3 eliminated all pain sense from the abdominal viscera (exclusive of the pelvic viscera). The study provides comparatively little information concerning the segmental innervation of the viscera but once the basic fact is established that visceral analgesia can be produced the refinements can follow. There is much to suggest, for example, that the greater splanchnic nerve plays a major role in the sensory pathway since histologically it contains a large percentage of afferent fibers and any minimal stimulation of the nerve (traction, crushing, faradism) in the unanesthetized patient causes severe and diffuse pain. Yet the study shows further that additional sympathectomy is necessary to denervate the ureter.

The balloon distention studies of the stomach and intestine in the normal are comparable in their results to those set down in Jones'[26] admirable monograph. Our postoperative balloon studies show that unilateral sympathectomy affects sensation of only the homolateral side of the colon while both sides appear to innervate the stomach and small intestine. There is a slight discrepancy between the findings in the small bowel after unilateral sympathectomy and those reported by Bentley and Smithwick[27]. The latter reported the elicited pain to be on the side of the midline away from the side of the

sympathectomy. This discrepancy is not so important, however, since the direct stimulation of the small bowel through the open abdomen after unilateral sympathectomy showed some tendency to this contra-lateralization of the pain.

Since the digestive tract is embryologically a midline structure it is not surprising that most of it appears to possess a bilateral sensory supply. It is more remarkable that the right and left parts of the colon appear to have lost their dual supply. The unusual finding of the apparent laterality of innervation of the lesser curvature of the stomach on intra-abdominal inspection is reported just as it was found; it may require additional study.

The lower end of the colon, i.e., the rectum, may have visceral sensory supply but like the oral end of the digestive tract the chief sensory supply seems to be somatic. White[21] has commented on similar findings of preservation of sensation in the lower colon following sympathectomy.

The intra-abdominal tests indicate convincingly that the pain sensitivity of the stomach and bowel is related to the mesentery or the visceral mesenteric junction and not to the remainder of the bowel wall. There has been conflict in the past on this point through the fact that Pacinian corpuscles exist almost wholly in the mesentery of the bowel had led many to doubt the existence of pain receptors except in the mesentery.

The line of demarcation anatomically between parietal and visceral peritoneum is not always certain and it has in places been arbitrarily drawn. The parietal peritoneum is usually presumed to be supplied by somatic afferents and this must be the case if one speaks of the ventral peritoneum because after bilateral sympathectomy this area possessed all its normal sensitivity. But the dorsal peritoneum overlying the head of the pancreas, for example, is also referred to anatomically as "parietal" in spite of the fact that it possesses Pacinian corpuscles[28] and loses its pain sensitivity after bilateral sympathectomy.

The appreciation of temperature in the stomach has been investigated by Wolf and Wolff[7]. Working with a man who had possessed a gastric fistula for many years they found that water in a 10 cm. bag in the stomach could be identified as hot and cold above or below 40° to 18° C., and beyond these extremes, variations of 3 degrees could be detected. The subject described the sensation as an area the size of his fist deep in the epigastrium but when the balloon was placed near the lower end of the esophagus a more intense sensation was elicited which was felt in the substernal area. Our observations tend to corroborate theirs and also to show that temperature sense (always closely allied with pain sense) is eliminated from the stomach by interruption of the sympathetics.

Past studies on sensation of the extra-hepatic biliary tract[29, 30, 31, 32, 33], as well as the present ones, show agreement in the area of constant reference of pain in the epigastrium. The occurrence of associated pain in the back in some subjects appears to be dependent on stimulation of the ducts, possibly at the sphincter of Oddi. The work of Davis, Pollock and Stone[11] shows that sensation in the gallbladder of dogs is supplied exclusively by the visceral

afferent nerves accompanying the sympathetics and more particularly the right splanchnic. Our studies in man show that the extra-hepatic ducts possess a bilateral sensory innervation although there were no observations on the results of distention of gallbladder after either right or left unilateral sympathectomy. Even though this item of information is lacking, it is fair to assume that in man the gallbladder too has a bilateral innervation and this assumes practical importance if nerve sections are to be put to use in the relief of pain.

The head of the pancreas has also been found in our studies to have a bilateral innervation. The importance of this finding was exemplified in a patient who had intractable pain from chronic calcareous pancreatitis of several years' standing. Her initial pain was in the mid-epigastrium and back. After right-sided resection of the splanchnics and ganglionated chain from the seventh thoracic to the third lumbar the former pain disappeared but pain of less degree now existed in the left hypochondrium. This left-sided pain was enhanced or duplicated by extra-abdominal pressure over any part of the palpable pancreas and in addition discretely localized stimulation (pressure and faradism) of the head of the pancreas after right sympathectomy caused left-sided pain. Following a left-sided sympathectomy all spontaneous pain ceased and no tenderness could be elicited on pressure over the previously tender gland (postoperative follow-up, 9 months).

The unilaterality of the sensory supply of the kidney and ureter from the evidence of these studies is definite and there is the additional suggestion that nerves other than the splanchnic play a role in sensory transmission at least for the ureter.

The investigations of Learmonth[25] led to his formulation of the triple sensory supply to the urinary bladder. Visceral afferents of the sympathetic system traverse the hypogastric nerves and plexus and the lumbar ganglionated chains. Visceral afferents of the parasympathetic system traverse the pelvic nerves which have their origins from the 2nd, 3rd and 4th sacral nerves. The somatic afferents traverse the pudendal nerves which originate from the 3rd and 4th sacral nerves. Our observations indicate that the interruption of any of the following nerve supply will not eliminate bladder sensation: bilateral lumbar sympathetics; parasympathetics, and pudendals (by section of all sacral nerve roots); bilateral lumbar sympathetics and both pudendal nerves. But interruption of all these will result in an insensitive bladder.

While the incentive to carry out these investigations was motivated primarily by a desire to learn something more about visceral sensation, some concern existed regarding the possible undesirable effects of the sympathectomies now being performed on so many patients with hypertensive cardiovascular disease. There is little doubt that after a bilateral thoracolumbar sympathectomy the visceral pain component of many abdominal diseases will be absent but in the most important ones (those, for example, that cause the acute surgical abdomen) there should always be other symptoms and signs. In two of our patients who had had bilateral thoracolumbar sympathectomy peptic ulcers perforated without there being any previous pain of the ulcer

but the patients vomited and showed all the signs of acute peritonitis which led to early diagnosis and successful operation. So far none of the several hundred sympathectomized patients of our series is known to have developed acute appendicitis, cholecystitis, or any other abdominal surgical disease. However, it is now our practice to perform cholecystography on most patients who are to undergo sympathectomy for hypertension; if gallstones are discovered cholecystectomy is advised unless otherwise contraindicated.

CONCLUSIONS

Studies of visceral sensation before and after sympathectomy in which the splanchnics and ganglionated chains are removed from Th_7 to L_3 on one or both sides show the following:

1. Pain sense in the stomach, intestine (except the rectum), extrahepatic biliary tract, pancreas, kidney, and ureter is mediated wholly by visceral afferent nerves which accompany sympathetic nerves within the area of the operative excision.

2. The kidney and ureter and the two sides of the colon have a homolateral sensory supply, but the remaining organs, with the possible exception of the gastric mesentery, have a bilateral supply.

3. Pain sensitivity exists not in the walls of the stomach and intestine but in the proximal mesenteries and at the mesenteric-visceral juncture.

4. Adequate stimuli for pain include, distention of hollow viscera; traction and faradic stimulation of stomach and intestinal mesenteries; manipulation, traction, faradic stimulation of the extrahepatic biliary tract, pancreas, kidney pedicle and ureter.

5. There is perception for extremes of temperature in the stomach; the afferent pathway accompanies the sympathetics.

6. The urinary bladder has a triple sensory supply via the visceral afferents of the sympathetic and sacral parasympathetic systems and the somatic pudendal nerves.

7. Interruption of appropriate sympathetic nerves may be employed for relief of intractable pain from some types of abdominal visceral disease.

8. The extensively sympathectomized patient due to the loss of visceral pain sense may undergo certain alterations in his response to visceral disease.

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DISCUSSION.—DR. LESTER R. DRAGSTEDT, Chicago: I should like to congratulate Drs. Ray and Neill on this interesting and, I think, very important contribution. Physiologists have felt for some time that the sense of hunger is due to contractions of the empty stomach.